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# DISEASES OF THE NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

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DISEASES of the NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

# Role of the Psychiatrist In the Rehabilitation of the Disabled Veteran

NEWMAN COHEN, M.D.
*Boston, Mass.*

It is generally recognized that one of the foremost challenges confronting this nation is the problem of the psychological, social, medical and vocational rehabilitation of war veterans with neuropsychiatric disorders. The process of rehabilitation may be defined as any therapeutic procedure directed toward the restoration of the individual to his most effective level. This process is carried beyond the relief of the specific condition until the person as a whole has attained the optimum use of his capacities.

In 1919, following World War I, we had but a meager understanding of the mechanism involved in behavior disorders. Available facilities for veterans care, in most instances, proved inadequate and were isolated from medical centers. Vocational schools were inadequately equipped and staffed and, although the value of vocational guidance was generally recognized, liaison among interested agencies was imperfectly coordinated. The problem of rehabilitation is essentially the same today as it was in 1919.

In order to approach intelligently the problem of vocational rehabilitation of the psychoneurotic dischargee it is necessary:

a. to recognize that veterans discharged for mental conditions were not necessarily insane. (Frequently this type of discharge indicated failure to adjust to military life);
b. to revise the mistaken attitude that most of these people are beyond salvage.

In most veterans hospitals vocational counselling is available when the veteran is prepared to consider entering the occupational domain. The veteran's appearance, attitude, intellectual endowment, aptitudes, abilities and interests are considered and explored, with the aid of objective test results, appropriately selected and interpreted, providing a supplementary means of evaluating accumulated data. These test scores should always be considered adjunctive to available psychiatric appraisals.

Personalized efforts at interesting the veteran in work are directed toward pragmatic vocational goals rather than mere mechanical distraction. A program selected by vocational appraisal should be effected in a setting as closely approximating actual working conditions as possible, its ultimate goal—a return to occupational usefulness. While work is not actually curative it nevertheless frequently assists in ameliorating and hastening the disappearance of abnormal functioning, thus facilitating the veteran's return to socialized and orderly adaptation. Lessons learned from the experiences of Army or Navy hospitals and reconditioning centers in successful reconditioning programs help the Veterans Administration do an outstanding job of rehabilitation.

Social service plays an important role in the rehabilitation process. On discharge from a hospital contact continues in an effort to aid and encourage the veteran to become interested in his new social milieu and readjustment to civilian life. This social infiltration is an important step in aiding

the war casualty and his family to a better understanding in overcoming barriers of indifference and prejudice. The job, his family and friends often determine whether he conforms or deviates from the mores of the group, and the community must be a distinctly supporting structure.

It is only through the combined efforts and the sincere and whole-hearted cooperation of veterans organizations, churches, the community, state, parents and social agencies working in conjunction with educators, psychiatrists, psychologists, personal counsellors and social service workers, that these veterans can regain their mental health, find a successful place in society, achieve happiness and gratification, and learn to face the future objectively and fearlessly.

### *Procurement of Employment and Vocational Rehabilitation*

The procurement of employment constitutes a factor of major importance in the overall rehabilitation process. Having suddenly re-entered competitive civilian life, war veterans were thrown on their own resources. Many employers adopted the attitude "Why should I hire a crippled or nervous man when I can secure a healthy or normal person," not realizing, or indifferent to, the harmful effects of such an attitude on a veteran's health. Although this attitude has lessened it still proves an obstacle with which we are forced to contend.

In the reorganization process, we face the problem of vocational training which must be effected in terms of the disabled veteran's needs and type of work for which he is best fitted and capable of performing. It is also important that the selected vocational objective prove commensurate with the veteran's intellectual and emotional assets.

Experience has shown that the success of the veteran's vocational training endeavor is strongly influenced by:

1. The type of mental disability.
2. His emotional reaction to disablement.
3. The suitability of his training objective.
4. The attitude of the employer and the training personnel.

Factors contributing to good vocational efficiency and productiveness are:

1. A previous well integrated personality.
2. A minimum of residual complaints.
3. An individual who is properly oriented towards his job.
4. A trainee who is capable of establishing stable interpersonal relationships.
5. A determination by the veteran to overcome the effects of disability.
6. The adoption of a realistic concern about the future.

Factors predictive of impaired industrial efficiency include:

1. The presence of residual complaints.
2. Personal liabilities including inferior intellectual endowment.
3. Troublesome personal problems, situational and domestic factors that distract attention from the job.
4. Attempts on the part of veteran to exploit their veteran status and demand preferential treatment.

Veterans placed in jobs above or below their mental capacities lose confidence and interest, consequently perform inadequately and usually quit, even though force of circumstances may have impelled them to accept the position. The resulting discontent may impair their morale and hinder further vocational attempts.

Personality factors must also be considered. How will the veteran adapt to his supervisors and fellow workers? Abnormal behavior patterns do not always imply a poor vocational prognosis for, if the individual properly performs his tasks, idiosyncrasies and shortcomings can be overlooked providing co-workers are not disturbed.

The importance of properly trained supervisory personnel in developing interpersonal relationships, thus obviating friction between employers and employees, cannot be overemphasized. Although an employer's prime interest is production he must exercise prudence and tolerance and should have some orientation along psychotherapeutic lines. If the employer or his representative is ever alert to unwholesome traits, attitudes, or emotional undercurrents, the issues can be met with an understanding and constructive approach mutually beneficial to employer and employee.

### *Vocational Problems with Specific Psychiatric Disabilities*

There must be a firm belief in the minds of employer and veteran, regardless of his handicap, that with a realistic appraisal of useable assets there is a place for the vet-

eran in the employment setting. Detailed information on the physical and emotional requirements of the job must be available. We must also recognize that in some cases readjustments entail seemingly insurmountable difficulties while in others rehabilitation can not be effected.

Difficult placement problems and sources of interference with occupational fitness may be included in the following group:

*1. Psychopathic Personality*

The term psychopathic personality though vague is nevertheless a convenient designation for a behavior pattern in which the following characteristics are prominent: A disregard for moral influences and conventional mores, rebelliousness, indifference to affection, rejection of authority and discipline and conflicts with the law. The psychopath has an undeveloped sense of responsibility, is impulsive and superficial in his emotional reactions. Members of this group usually have a history of poor vocational adjustment and repeated failure to attain a socially acceptable status.

From the standpoint of psychiatric treatment it is generally assumed that little can be accomplished for these individuals. Many do not want help and will prove a source of trouble wherever they are found. These veteran patients with psychopathic personality traits would have difficulty in effecting an adult type of adjustment whether or not they served in the armed forces. However, frequently these people are of above average intelligence whose potential energies might be gainfully utilized in a controlled vocational setting. These patients must be appraised in terms of assets and liabilities which have prognostic value for vocational adjustment. The following is a case example of such a type.

Carl, age 24, is such a problem. An illegitimate child abandoned at two weeks of age and again unwanted when his foster parents, financially able to give him every advantage, separated, he was shuttled among various people who were at times hostile or only superficially interested in his welfare. He ran away from home at 13, wandered about the country and supported himself by doing odd jobs. His lack of moral foundation coupled with premature physical development resulted in extensive amoral experiences. Returning "home" for a brief and stormy period he was drafted at 19 years of age into the Navy, followed by the harrowing experience of Pearl Harbor, later sustaining a fall in which he landed on his head. Clinical findings were negative but a year later an acute psychotic reaction developed which lasted two months and resulted in discharge from the service. He again returned home but on initiating a life of moral looseness he was driven out. He again supported himself performing odd jobs and when convenient formed casual relationships with prostitutes, homosexuals and other bohemian types. Although constantly in trouble he evaded any serious charge and was eventually brought to a mental hospital by the police. Found in a state of collapse one morning, he related a fantastic story resulting in his commitment but was shortly discharged as a constitutional psychopath and referred to the Veterans Administration Rehabilitation Section. Following a careful psychiatric consultation he was referred to a vocational counsellor for determination of his objective potentialities, apart from his own hopes, fantasies and fabrications. A review of past vocational attempts, supported by psychometric test results, indicated a fighting chance to effect an adjustment, if given appropriate opportunity and continued guidance. Following a course of psychotherapy at the Mental Hygiene Clinic and vocational counselling, Carl with the concurrence of the Psychiatric Consultant and the Training Officer, was enrolled at a local dramatic school with concurrent evening study for completion of high school work. His income being limited to his small pension and subsistence allowance, he could make ends meet but there was nothing for unnecessary frills. At first dubious progress was reported but following a few stormy sessions with the Training Officer, the veteran's program was adjusted with the cooperation of the school faculty and, although still egocentric and demanding Carl enjoys the recognition he is receiving, accepts the disciplining and likes the school. He has appeared professionally on the radio, has been selected for a coveted dramatic trophy and, although not rehabilitated, does have an objective and a maturing approach to his problem. Aware that the road ahead is difficult he accepts the challenge in a constructive manner.

*2. Post Traumatic Reactions*

This category comprises those patients who have sustained some cranial injury. The trauma may be either physical or psychological although patients with gross neurological changes are usually considered in this group. The symptoms are not unlike those seen in the conventional anxiety states. There may be transitional cases presenting characteristics of an organic and a functional condition, hence a differential diagnosis is difficult. Among veterans with post concussion states seen by the writer, headache, dizziness, fatiguability and anxiety are

predominant symptoms. When the neurological and laboratory symptoms are negative, there is the possibility that we are dealing with psychically conditioned symptoms. A constructive approach to the problem of post traumatic states should include:

a. a physical examination; b. prophylactic measures; c. psychotherapy; d. vocational training.

A recent referral, showing many typical manifestations of this disorder, is John, 24 years old, married and with one child. His history indicates he was a sensitive emotional child reared in an over-protected and solicitous atmosphere, who graduated from high school and worked but a few months at unskilled labor until drafted. As a Marine he had extensive combat experience, was exposed to a number of near misses finally receiving a glancing blow to the head, with relatively light injury. Although he lost consciousness after an intense explosion the episode was not reported and no overt signs of emotional difficulty became evident until approximately eight months following his discharge from the service. He developed many neurotic symptoms which appeared to revolve around his head injury and applied to Veterans Administration for assistance. Apprehensive, jumpy and tremulous, with hyperhidrosis prominent, he complained of dizzy spells, feared he was going insane and that he might commit an act of violence. His uncontrollable fear and panic states were completely disabling and he experienced occupational placement difficulties. An organic basis for the disability was excluded by physical examination, electroencephalogram and laboratory studies. Obviously suffering an emotional disability he needed guidance, encouragement and direction. Psychotherapy and vocational counselling were initiated and business college training, with concurrent psychotherapy, was agreed upon. This has recently been completed, the veteran graduating with honors, and on the job training as an accountant is now in progress under supervision of an understanding Training Officer. Although the veteran is still somewhat tense and of slightly agitated mood, symptoms have decreased or disappeared. His employers are aware of his difficulties and permit an emotional leeway which it is anticipated will soon be unnecessary. This is another instance of pragmatic approach toward the rehabilitation of a disabled veteran in which psychotherapy and gainful training toward a psychologically satisfying objective went a long way towards allaying the veteran's anxiety, restlessness and neurotic symptoms.

### *3. Alcoholism*

The complicated problem of alcoholism, which has long been regarded as simply a moral problem, is a challenge to the process of rehabilitation. It's inherent and complex factors merit scientific scrutiny and therapeutic attacks, for the modern conception regards alcoholism as a symptom of underlying diseases. Alcohol is employed much in the manner of psychoneurotic symptoms to satisfy inner needs, relieve anxiety and tension and as a means of blurring the sharp outlines of reality and cushioning its blows. These poorly organized personalities lack self-control and show impairment in psychological functioning, resulting in physical harm and interference with psychosocial adjustment. The alcoholic addict rarely directs his efforts toward overcoming his habit, seeking the pleasures of the senses and scorning boredom, unpleasantness and obligations. He has adjustment problems stemming from the conflict between desire to drink and the taboo of social convention. Another problem is the paradoxical attitude of society which is amused at the alcoholic because of his mirth provoking behavior, but if the "funny man's" behavior becomes offensive, censures and punishes him.

Drawbacks to the successful rehabilitation of alcoholics include:

a. failure of the patient to cooperate or lack of desire to be cured; b. society's insufficient interest and negativistic attitude toward the problem; c. lack of proper treatment facilities.

Treatment has largely been provided by law enforcement agencies and has consisted of confinement in penal institutions. Prominent among the great variety of treatments is the conversion method of "Alcoholics Anonymous" and the aversion conditional reflex method. Davis[1] stresses the importance of recreation as a therapeutic measure. Treatment efforts also include physical exercise, resocialization, and occupational therapy. Favorable results have also been obtained with subshock doses of insulin.[2,3] Supportive treatment, redirection of goals and the attainment of insight also aid in combatting a return to addiction and reorienting the patient to a socially acceptable manner of living. Treatment in a semi-hospital setting appears to be a progressive and constructive approach to the problem. The psychiatrist, psychologist, vocational and personnel counsellor and psychiatric social

worker aid in determining sources of inadequacies and the patient's rehabilitation.

*4. Epilepsy*

If each case of epilepsy is earnestly appraised in terms of the particular situation, the vast majority of this group can be gainfully employed without danger to themselves or others. According to Cobb and Lennox[4] 51 percent are fully able to work, 28 percent are partially able, 18 percent are unable because of seizures and 3 percent unable for other reasons.

Although epileptic attacks tend to diminish in frequency when the individual is employed or occupied, work involving excessive strain or stress should be avoided. Epileptics should not be placed in jobs where the safety of themselves or others is imperiled through "blackouts" or uncontrollable seizures. Generally, mechanical and electrical hazards, exposure to moving vehicles or burns and jobs involving working at altitudes should be avoided.

A fundamental problem the epileptic faces when seeking employment is the barrier occasioned by the employer's bias and prejudice. The employers objections to hiring epileptics are usually due to either misconceptions about epilepsy or the compensation laws which make the employer liable for injuries sustained by employees while working. We must counteract the belief that epilepsy denotes something sinister. The stress of hard work is not harmful to those subject to seizures, in fact attacks are most likely to occur when the epileptic is idle or asleep, and in the majority of cases activity of the brain and muscle proves an important antagonist of seizures. It is false to suppose that epileptics are defective in mind, body or personality. As a consequence of these conditions a person who has seizures, particularly those veterans unable to secure gainful employment, must conceal the disability. This means that neither he nor the employer can utilize safeguards against injury which might be used if the true state of affairs were known.[4]

Rehabilitation of epileptics is aided by:

a. treatment for seizures; b. vocational adjustment, and c. supportive measures to encourage normal active life and an appropriate philosophy regarding their illness.

*5. Psychotics*

Shock therapies have often produced spectacular results with no impairment of intellect or emotion, enabling veterans suffering from a psychosis to return to normal living and without need for new job orientation, especially if they adjusted satisfactorily on their last job. However, some veterans with schizophrenia or brain damage may have need for a new job consistent with their limitations and though the tempo of their work may be reduced they often retain previously acquired knowledge.

Psychotic cases should be considered from a multi-dimensional point of view. In the treatment of schizophrenia occupational therapy, which may be likened to collective psychotherapy, serves as a spiritual, psychological and physiological stimulant. Its effects impinge on the personality, counteract unfavorable clinical aspects, accentuate the individual's personality aspects, activate and strengthen normal functioning, frequently negating the psychic and emotional pathology. Hospital care is generally required in the severe cases but among these patients are workers capable of superior performance in almost any type of work and, following remission of the psychotic process, employment is possible and should be expedited at the level of activity the veteran can enjoy, for its educational and psychotherapeutic effect. Generally work has beneficial effect on the introverted pre-occupied schizophrenic, helping to bind him more closely to reality, somewhat counteracting unfavorable clinical aspects of his personality and deters him from a state of idleness and indifference. Recourse to occupational therapy may find something that will arouse his interest, enabling him to forge ahead toward gainful employment.

*6. Mental Defectives*

Attempts were made at induction stations to eliminate this group from the armed forces, and relatively few are found in the veteran population. Although members of this group are incapable of competing with normal individuals and rate low in economic efficiency they are capable of performing

simple unskilled routine and repetitious types of manual work and may attain satisfactory economic and occupational levels.

Generally sensitive of the fact that he is not the equal of others, the mental defective needs a job where the required skills are not beyond his capacity and where there will be a feeling of responsibility on the part of his employer for extra guidance, repetitive training and praise for achievement. Psychological tests are valuable in determining vocational potentialities but must be cautiously interpreted and evaluated in terms of psychiatric appraisal and work performance. Veterans of this group have no hope of complete recovery and the problem is essentially one of making the most of imperfect material.

*7. Blindness*

Psychological adjustment to this handicap constitutes a problem of magnitude as the psychosociological determinants may prove more devastating and destructive than the actual physical handicap. The rationale behind the rehabilitation process is to keep these veterans hopeful, cooperative and to effect the role of an independent adult. While guidance and counselling are helpful it is of prime importance for the blinded individual to take a realistic personal inventory of himself and that his abilities and strivings are accorded an outlet and receive proper recognition. Most states have vocational rehabilitation advisers experienced in counseling and placement of the blind veteran, however, places to train these veterans in self care are urgently needed.

*8. Deafness*

This handicap also constitutes a rehabilitation problem of magnitude as the disability places an individual at tremendous disadvantage and often plays havoc with his attempts at social and economic adjustment. The World Wars demonstrated with much prodigality of example that deafness may represent a conversion phenomenon. Defective hearing is frequently over-shadowed by such resultant disturbances as auditory hallucinations and paranoid reactions, which often prove more incapacitating in the veteran's psycho-social and economic readjustment than the disability itself. Additional patterns of reaction frequently characterizing the disturbed personality of the deafened include: suspiciousness, sensitiveness, social anxiety and aggressive behavior. Deafness is many times employed as a mechanism to obtain a silent environment in the same manner that disturbance of vision may represent a symbolic desire to avoid unpleasant sights.

Normal hearing cannot be restored by means of a hearing aid but by its use it is possible to approximate this goal. Hence these individuals should be taught how to use such an aid, supplemented by instruction in lip reading for, unless the individual understands the limitations of a hearing aid and realizes it will not prove a panacea for his difficulties, he is apt to react with confusion and disappointment even discarding the hearing device entirely. Psychotherapy has proven effective in promoting a wholesome perspective and in the reconditioning program efforts are best directed at inculcating socialized tendencies and at channelling the individual's interests into stimulating and constructive pursuits. In the matter of vocational placement consideration should be given to occupations not primarily demanding dependence on the spoken voice. Unfortunately, industry's concept of what the deaf and blind can do is low.

*9. Deformity, Disease, or Injury*

The scope of these problems includes amputations, paralyses, brain and cord pathology, disfigurement and orthopedic deformities. These veterans should not be regarded as poor risks for handicapped workers, grateful for their gainful employment often prove loyal and excellent workers. Frustration can be reduced by early placement with emphasis on the capacities and potentialities rather than the handicaps of these individuals. In determining how a handicapped veteran can fit into the overall readjustment process, the following factors are considered and realistically appraised:

a. his work record prior to disablement; b. the physical capacities which can be utilized in worthwhile production; c. the physical and emotional requirements of the job.

*Discussion and Summary*

Many types of mental disorder seen in military life did not differ materially from

those occurring in civilian life and breakdowns in personality were frequently predicated on the equipment an individual carried into military service. Induction necessitated a solution of the strong emotional conflict between the instinct of self-preservation and the equally powerful sense of duty and attempts to lessen the friction of these emotional cross currents often occasioned a pathological compromise and subsequent difficulties.

Factors considered conducive to the development of psychic conflict include prolonged exposure to stress of combats, mental and physical fatigue, poor adaptation to comrades, loneliness for home and regimentation associated with military life. When these demands exceeded the soldier's threshold of resistance his personality organization became disordered.

The number of discharges because of mental disabilities reached enormous proportions during World War II. "Over a third of the battle casualties were neuropsychiatric."[5] Approximately every other bed in veterans hospitals is occupied by a mental patient. Intelligent care, sensible guidance and understanding help forestall undesirable or abnormal behavior patterns and expedite the veteran's return to a normal mode of living.

The rehabilitation program was established to assist the disabled veteran in his readjustment to civilian life. Its guiding purpose is to prevent dependency, promote a spirit of cooperation, and to treat the veteran as a sound, upright, independent and mature individual. A constructive program should embody: a. medical diagnosis and appropriate treatment; b. vocational guidance and training directed towards a definite vocational objective; c. procurement of employment and follow-up.

The rationale of rehabilitation consists of the restoration of an individual to his highest functional level of attainment socially, culturally and economically, and while work is not of necessity a curative agent its therapeutic value in hastening the veteran's adjustment cannot be overemphasized. There should be a close working arrangement between rehabilitation and employment agencies.

In attempting to meet our obligations to the disabled veteran we should be guided by the thought that:

1. The payment of a premium (compensation) to veterans suffering from neuropsychiatric disorders, does not in itself fulfill our obligation to them.

2. Effective vocational rehabilitation includes the concerted efforts of adequate treatment facilities such as the Mental Hygiene Clinic, to which the neurotic veteran may be referred for psychotherapy prior to vocational training or on a concurrent basis as the individual case warrants.

3. The vocational rehabilitation of veterans suffering with neuropsychiatric disabilities calls for a more sympathetic, tolerant, understanding approach than that ordinarily required with the physically handicapped veteran.

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# Vibratory Sense - A Non Specific Sensory Modality

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The surgical procedure of antero-lateral chordotomy introduced by Spiller and Martin[1] in 1912 and since reported by numerous authors has greatly enhanced the study of the spinal cord in man under relatively normal conditions. The sensory changes reported following antero-lateral chordotomy have mainly been concerned with contralateral loss of pain and temperature. I have observed 3 instances in two patients in whom vibratory sensation has been diminished on the contralateral side at a level corresponding somewhat to that of pain and temperature following this procedure.

The pathways of vibration sense are not entirely agreed upon, nor have the anatomical effects following chordotomy been consistent. Studies of the peripheral receptors of vibratory sensation have led mainly to two opposing points of view. The majority of writers[2,3,4,5,6,7] have felt that vibration sense is dependent upon the superficial and deep tactile receptors while Katz[8] (1930) believed there are special receptors for vibration sense. Some of these authors[5,7] believe that vibration should not even be thought of as a specific sensory modality, but rather as the sensation resulting from rapid repetitive stimuli of touch or proprioceptive endings. This sensibility has been considered by most authors to be carried by the ascending pathways in the posterior columns of the spinal cord, while a few[7,9] believe that some of the vibratory fibers are carried elsewhere.

I believe that this report will contribute some support to those who state that vibration sense is also carried by ascending tracts other than those of the posterior columns, and that it is dependent upon the superficial and deep receptors and not special receptors. The following are brief case reports showing contralateral diminution of vibratory sensibility following antero-lateral chordotomy at an approximate level corresponding to pain and temperature loss.

Patient A.L.M., 36 year old colored female, was admitted to John Sealy Hospital May 19, 1950 for intractable pain of her right lower extremity. Her pain followed a severe traumatic injury in September of 1949 to thoraco lumbar region of the spinal cord. The only abnormal neurological findings at the time of admission were diminution of pin prick over the lumbar 4, 5, and sacral 1 Keegan dermatomes on the right, and weakness of both lower extremities. A lower cervical antero-lateral spinal tract section was performed on May 24, 1950. Following the operation and at a five months follow up, this patient had a sensory level at about thoracic 10 for pain. Temperature and light touch were abolished one to two segments below. Vibratory sense was diminished at the pain level on the right side. Position sense was intact.

Patient V.L., 34 year old white female, was admitted to John Sealy Hospital July 22, 1950 for relief of intractable pain due to a squamous cell carcinoma of the cervix. There were no abnormal neurological findings. On August 21, 1950, a mid cervical antero-lateral spinal tract section was performed. After this operation, this patient had a sensory level for pain at about T 2 and one for vibration and temperature about 2 inches lower. The vibratory level was not one of complete loss, but rather of diminution as compared to above the level and on the other side. Then on August 29, 1950, a left upper thoracic antero-lateral spinal tract was carried out. Following this procedure the patient had a sensory level on the right side at the costal margin for pain and an inch below for temperature. Light touch was diminished at a level two segments below. Position sense was intact. Diminution of vibratory sense was found at a level corresponding to that of temperature. (Figure 1.)

Diminution of vibratory sensibilities on the contralateral side of the body at a level roughly corresponding to the pain and temperature levels was well demonstrated in these two patients following antero-lateral spinal cord section for intractable pain. Even though the method of examination by the use of a tuning fork is a very crude one, I believe that these results would substantiate themselves with a more sensitive test. The portion of the cord that is sectioned in this procedure is shown in figure 2. The knife enters the cord just anterior to the dentate ligament and is then carried anteromedially about 4 mm. and brought out just lateral to

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*Figure 1.* Patient V. L. Sensory levels outlined after bilateral antero lateral spinal tract section. Vibratory sense and temperature affected at same level on the contralateral side of the chordotomy.

the level of the anterior roots. The more perpendicular the cut of the spinal cord at the plane of the dentate ligament, the higher the sensory level obtained.

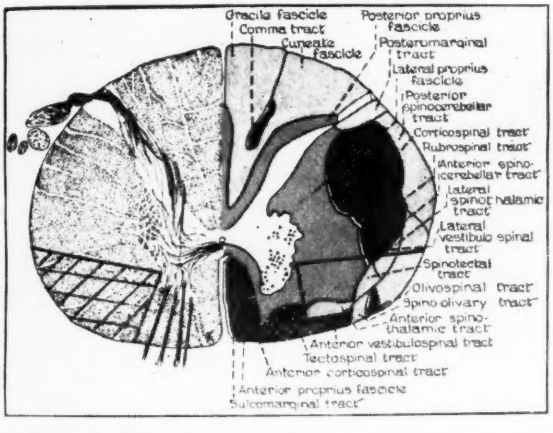


*Figure 2.* The cross hatched area represents the spinal cord sectioned in the two patients reported. Diagram taken from Larsell's Textbook of Neuroanatomy, 1939. D. Appleton-Century.

In an anterior-lateral chordotomy the following ascending tracts might be severed: dorsal spinothalmic tract, ventral spinocerebellar tract, spinotectal tract, spinal olivary tract, sulcomarginal tract, and the anterior spinothalmic tract. The tracts we might be concerned with here would be those made up of crossed fibers which include the anterior and dorsal spinothalamic, spinotectal, and ventral spinocerebellar tracts. The only sensory modalities carried by the dorsal spinothalamic tract are pain and temperature. Stookey[10] reported that even these two sensibilities can be separated within the tract. Sherman and Arieff[12] reported patients with spinal cord disease exhibiting dissociation between temperature and pain. However, most investigators believe that in general these fibers are greatly mixed with each other. Little is known as to function of the spinotectal tract, but it is considered a correlation pathway. The ventral spinocerebellar tract carries proprioceptive impulses to the cerebellum of which part are contralatera.

The ventral spinothalamic tract is considered to convey light touch and pressure sensations. Strong and Elwyn[9] state that this tract supplements the tactile path of the dorsal white column. In injury there is a heightened threshold effecting a reduction of touch and pressure points per unit of skin area.[13]

Weinstein and Bender[14] have found that with tumors of the thoracic or lumbar region of the cord, vibration sense was impaired before position sense and to a greater degree, while in neoplasms affecting the brain stem and upper cervical region the converse was true. They thought that such dissociation may be due to the fibers of the posterior column at the cervical region travelling to both the thalamus and cerebellum, while the fibers of the lumbar and thoracic regions travel via the medial lemniscus. Hamilton and Nixon[15] in reporting the sensory changes of pernicious anemia stated that there may be a dissociation of vibration and position sensibilities and explained the possibility of these two modalities being sufficiently separated within the posterior columns, or that the fibers subserving joint sensibility may pass up the cord in two pathways. It would seem from the present material that the fibers subserving vibration sense may pass up the cord in two pathways.

Touch was diminished on the contralateral side in these two patients. Walker[16] stated that touch was not entirely spared following anterolateral chordotomy in that the number of touch points per square cm. are reduced. This reduction was observed by him as occurring bilaterally and therefore indicated bilateral origin of fibers of the anterolateral column. According to Larsell[17] lesions below the cervical region of the cord if confined to one side have little or no effect on touch and pressure sensations indicating a double pathway—one through the posterior funiculus and the other through the lateral funiculus. Hyndman and Van Epps[18] in numerous deep sections extending to the anterior median fissure observed no deficit in perception of light touch, deep pressure, two point discrimination, position sense or vibration sense. The associated contralateral diminution of touch would give some support to the thesis that vibratory sense is then dependent to some degree on the intact touch fibers.

It is therefore rather difficult to assess a single ascending tract in the anterior lateral column with the transmission of vibration, but it seems that the ventral spinal thalamic tract might assume most of this responsibility, especially with the contralateral loss of touch, or one might suspect a supplementary tract as the conveyor.

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# Carbon Dioxide in Obstetrics

(A Preliminary Report)

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The drama of pregnancy is beset by a complexity of hopes, fears, joys and self-consciousness, climaxed by the ordeal of bringing a new baby into the world. Much has been said and written about painless childbirth but the major threat has been that in saving pain for the mother perhaps the baby will suffer. A variety of analgesics and anesthetics has been used during labor but they all have some drawbacks. As a result there has been a trend in some quarters to try to do without them almost entirely, to rely instead on psychological preparation, relaxation and acceptance that some pain is necessary but that it is not nearly as terrible when the woman's mental attitude is right.

For a period of three years the author has been using carbon dioxide comas* in the treatment of a variety of psychosomatic and psychoneurotic problems in private practice. A mixture of 20% $CO_2$-80% $O_2$ or 30% $CO_2$-70% $O_2$ is used for the first ten breaths and then the $CO_2$ percentage is gradually increased til coma or sleep is induced. Inevitably, pregnant women came in for relief of their emotional problems associated with their pregnancies. It was with some trepidation that the author undertook to treat these women with "$CO_2$" because the physiological response includes a period of forceful breathing and the abreactions sometimes include vigorous motor responses such as kicking, twisting and straining. The ever-present question was, "Would these treatments cause miscarriages and if so, at what period of pregnancy would this be a danger?" It came as a pleasant observation that the occurrence of miscarriage was less than in the preceding several years of managing a considerable number of pregnancies in private general practice. A percentage of these women had been in such turmoil about their pregnancies that they were tempted to try to end the pregnancy prematurely and it was found that, aided by the "$CO_2$'s," they were able to face their pregnancies better and were more reconciled to their life situations. The conclusion from these observations was that the physiological and psychological reactions to the "$CO_2$'s" not only did not endanger the pregnancy at any period, but actually improved the mental attitudes and the physiochemical responses to the pregnant state.

Inasmuch as a "$CO_2$" coma is also a brief anesthetic, it gradually came to mind that it might be used during labor itself in the place of other anesthetics. The main barrier to the general use of "$CO_2$" comas as an anesthetic during labor is that the abreactions to the "$CO_2$'s" may appear to the inexperienced doctor or observer as distressing and unpleasant. Naturally, if these abreactions have been worked out before actual delivery the procedure at that time is smoother, but even if "$CO_2$" is first introduced to the patient at the time of delivery the reactions are beneficial, even though at times dramatic.

The mother needs aid in childbirth in handling three types of feelings. Firstly, the feeling of pain is present in varying amounts. This pain is a natural accompaniment of delivery and is not nearly as important as one would think. Secondly, the feelings of conscious fear, which are on the basis of the real dangers to both mother and child, are best handled by reassurance and intelligent explanations. Thirdly, there are the unconscious fears, those feelings of which the patient is not aware but which cause changes in pulse rate, respiratory excursions, uterine tone, uterine contractions and many other body changes. Unconscious fears may be called feelings because, even though usually they cannot be felt, after

*For simplicity of reference, "$CO_2$" is used in this article to mean a suitable amount of a mixture of carbon dioxide and oxygen to induce coma or sleep.

"$CO_2$" treatments the patient does become aware that they are the causes of these body changes. As these unconscious feelings are raised to consciousness they have less physiological effect on the patient.

During this investigation, in some cases the initial carbon dioxide treatments were given during the early months of pregnancy; in others, at regular intervals during pregnancy, about five treatments per patient; in still other cases, the patient was seen for the first time at delivery. During pregnancy the "$CO_2$" treatments were helpful in controlling the vomiting of pregnancy, insomnia or unusual nervousness. Right after a treatment the patients often would be able to tell the doctor what they were afraid of, such as fear of their husbands leaving them, hemorrhage, their in-laws, or losing the baby. Before the treatment they themselves often would not know what they feared.

This study includes thirty-one women delivered in the home. In the home deliveries the author now uses "$CO_2$" almost exclusively. Of these delivered in the hospital, due to hospital rules, none had "$CO_2$" on delivery. It appeared that those who have had "$CO_2$'s" during pregnancy and were delivered in the hospital, were much easier to handle than the ones who have not had "$CO_2$" treatments during pregnancy. Of the thirty-one patients delivered in the home, seven did not have "$CO_2$" at the time of delivery as they delivered before the doctor arrived. This reflects the lessened anxiety and panic in the patients who had their tensions previously decreased by "$CO_2$" inhalations. Three who were delivered in the home had ether for the actual delivery. They are included in this series as they were given the "$CO_2$" in adequate amounts during pregnancy but at the time the author had not yet decided to try it at delivery.

Twenty-one cases had "$CO_2$" as the anesthetic at delivery. A few were given a few inhalations of ether or chloroform to see which they preferred and all but one asked for the carbon dioxide. Some women had demerol and several had barbiturates but the majority got along on carbon dioxide alone.

Various techniques were used in administering the "$CO_2$." Giving a few breaths first and draining off the abreactions a little at a time until the patient was able to take the inhalations easily without abreactions, worked well if there was sufficient time. One can give three to five breaths of the 30% $CO_2$-70% $O_2$ mixture to those patients who are not too nervous, however, if the patient is nervous it is better to give a couple of "$CO_2$" comas about fifteen minutes apart. Having thus drained off the nervous reactions, the doctor can then proceed by giving three to five breaths of the mixture with each pain and finally again induce coma at the moment of delivery. An assistant can easily handle the actual administration under the doctor's direction. The margin of safety is very large. It is necessary to use some other type of anesthesia for repair as the patient wakes up in two minutes after the carbon dioxide is stopped. During the delivery, as "$CO_2$" is given, the contractions of the uterus increase in effectiveness, and do not decrease as is occasionally experienced with ether. If the mother is dozing or sleeping from barbiturates or demerol, the carbon dioxide tends to awaken her. With "$CO_2$" alone the patient can be completely awake with a clear head in two minutes and intelligently discuss the situation. The babies were born more pink than usual. There was not one blue baby in the lot. No mother or baby in this series showed evidence of any morbidity due to the carbon dioxide. The author considers that there is no contra-indication to the proper use of carbon dioxide as an anesthetic in obstetrics. Obviously various physical disabilities such as active tuberculosis, decompensating heart disease, and eclampsia present problems of management in the use of any anesthetic and caution is wise. The present evidence is that the "$CO_2$" is less hazardous both for mother and baby than other anesthetics.

### *Conclusion*

Inhalation of suitable mixtures of carbon dioxide and oxygen, producing coma, has been found both safe and beneficial in treating pregnant women at all stages of their pregnancies. As an anesthetic for delivery, "$CO_2$" coma is more satisfactory both for the mother and baby than other anesthetics.

The abreactions to this procedure need to be understood and utilized by the doctor. Some other anesthetic is needed for surgical repairs because the "$CO_2$" coma is brief.

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# The Sentinel Asleep On Post

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The sentinel who falls asleep presents a problem in cerebral physiology as well as in military discipline. In 1943 a study was made of 25 soldiers who had been found asleep on post, and the object of this paper is to give the results of the study and to consider the physiology of sleep insofar as it relates to the special problems of the sentinel. It is hoped that the results will be of interest to court martial and reviewing authorities, who must assess the culpability of the defendant, and to medical officers, who must understand why a man falls asleep when he shouldn't.

The men fell into three groups, which are listed in order of culpability.

GROUP I.—Four men who fell asleep because of insubordination. They were rebellious and did not want to obey orders.

GROUP *II.*—Nineteen men who fell asleep because of apparent carelessness.

GROUP III.—Two men who fell asleep because of narcolepsy.

*Group I—Insubordination*

The four men in this group were rebellious psychopaths, with a previous history of truancy and delinquency. In the Army they had been flagrantly insubordinate. In falling asleep on post it was clear that they had not succumbed innocently, but, on the contrary, had taken every possible means to encourage sleep. Thus one man had parked himself on the back seat of an Army staff car where he was found asleep with tie undone, shirt unbuttoned and shoes unlaced. Another had stood his rifle against a wall and had crawled under a building where he had spread his raincoat and lain down on it.

*Group II—Carelessness*

The nineteen men in this group had no conscious desire to disobey orders, but fell asleep rather because of accidental circumstances, combined in some instances with a measure of carelessness. To be sure, the seeming accident or careless slip may really be the expression of unconscious drives, a fact which must be kept in mind in the analysis of any apparently accidental infraction.

The men were, in the main, good soldiers, and some of them had earned high praise from their superiors. Most of them fell asleep because for one reason or another they had gone on post insufficiently rested. Sometimes a man just didn't have the foresight to get his sleep in good time, going to town instead for a few drinks or a movie. In some cases there were factors beyond the soldier's control. Thus, one man who stood guard at night was housed next door to the regimental band, which never failed to start practice in the morning at eight o'clock sharp. In some cases the offenses had taken place in the summer, and the men complained that in mid-day it had been too hot to sleep.

The following story is typical. The soldier, age 23, a man in good health and of pleasant personality, wanted to have some time off on Monday, so he traded guard shifts with a friend. On Tuesday, from midnight to 4 a.m. he served his friend's shift, and from 4 to 8 his own. The rest of the morning he had breakfast, cleaned his rifle and stood guard mount. From noon to 4 p.m. he was on post again. He then tried to get some sleep in his tent, but it was a hot day in July and he slept only two hours. He

went on post again at midnight. It was customary for the night sentinel to turn off the radio for one of the sergeants who usually fell asleep with it on. At 12:45 a.m. (Wednesday) the sentinel went into the sergeant's tent, found him still awake, and sat on a cot and talked with him. The sergeant fell asleep, and the sentinel lit a cigarette, which is the last thing he remembers. He was found asleep an hour later, sprawled on the cot, fully clothed, his rifle beside him. Sleep evidently had overtaken him unawares.

The contrast between Groups I and II, in respect of the soldier's attitude and demeanor is markedly apparent. The men in Group I were hostile psychopaths; resentment and rebellion were written on their faces. Some of them, in spite of the plainest evidence, insisted with a straight face that they had not been asleep as charged. Others admitted that they had, but argued that for one reason or another they should not have been posted as a sentinel. By contrast, those in Group II were men of agreeable disposition and, on the whole, had adapted well to the service. Having been caught off base, they freely admitted their guilt and were eager to redeem themselves.

### *Group III—Narcolepsy*

Narcolepsy is the commonest of the chronic diseases that cause somnolence. It is manifested by sleep attacks and attacks of paralysis. It was comparatively unknown prior to World War I, when the military aspects of sleepiness attracted attention to it.

As a rare disease, narcolepsy is not very important in its own right, even if its victims do sometimes get into trouble because of it. But it is tremendously important for the light it sheds on normal physiology. Thus, to take but two examples, the study of narcolepsy and its physiology has revealed why soldiers get sleepy at the front line, and why athletes feel sleepy before the start of a race. Moreover it has shed light on problems other than sleep, notably on fatigue.

The key to the physiology of narcolepsy may be found in Pavlov's work on cerebral inhibition.[2] Without using drugs, and merely by manipulating the environment so as to evoke "internal inhibition," Pavlov caused some of his animals to fall asleep. This epoch-making discovery (which has nothing to do with animal hypnotism) led Pavlov to the conclusion that sleep and inhibition are one and the same thing, sleep occurring when inhibition has irradiated sufficiently to involve all or nearly all of the cerebral cortex. The bearing of this work on human physiology has already been reviewed.[3]

Of the four methods which Pavlov used to produce inhibition—experimental extinction, conditioned inhibition, delay and differentiation—the second and fourth have a direct bearing on the physiology of sentinel duty and the remainder of the paper will be devoted to these.

### *Conditioned Inhibition*

Having established a conditioned reflex, Pavlov then modified the experiment by omitting reinforcement whenever the conditioned stimulus was accompanied by a second stimulus. For example, in the case of an animal trained to expect meat after the sounding of a bell, a new experiment was now started: the bell was now sometimes accompanied by a flash of light, and meat was offered only after bell alone, never after the combined stimuli, bell plus light. At first the animal, not appreciating the significance of the additional stimulus, light, salivated in response to the combination. With proper training, however, he learned to differentiate the combination, bell plus light, from bell alone, and he no longer salivated in response to the combination, but continued to do so to bell alone. Pavlov showed that the combination, bell plus light, did not fail to evoke excitations in cerebral salivary centers, but that these excitations were held in check or inhibited. He demonstrated this by presenting an alien stimulus a few moments after the combination, whereupon salivation, till then inhibited, appeared in full force. Pavlov spoke of the "positive stimulus" (in the example given, bell), the "negative or inhibitory stimulus" (light), and the "inhibitory combination" of the two.

In experiments with conditioned inhibition, as in those with the other three methods, some animals responded to the inhibitory combination by falling asleep. It was

this discovery that led Pavlov to conclude that inhibition and sleep are one.

The bearing of this experiment on man lies in the fact that conditioned inhibition occurs in man too, and that it may put man to sleep just as it does the experimental animal.

Whenever man suppresses a forbidden impulse, there is conditioned inhibition. An impulse may be permissible under some circumstances, but forbidden when other factors are added to the situation. An example is furnished by the soldier at the front line. The normal response to danger is to run away from it. This is what civilians do when dynamite is about to go off. But the soldier at the front recognizes an additional factor which alters the situation, a factor which requires him to stay at his post whatever the cost. His impulse to run away is held in check by conditioned inhibition.

That soldiers at the front tend to get unnaturally sleepy has been observed in both great wars. MacCurdy[1] in World War I noted that soldiers moved up to the front and exposed to shelling for the first time reacted in some cases with "lethargy so extreme that the individual will lie down and go perforce to sleep with a pathological indifference to the danger." This sleepiness is the more striking for having been displayed by soldiers in their first experience of enemy fire—a moment when they might have been expected to break all records for alertness. The same reaction is seen in civilian life. Narcoleptic workmen sometimes fall asleep in situations of danger, as on a scaffold, where the impulse to escape must be suppressed because there is a job to be done.

One may wonder that in the suppression of an impulse enough inhibition may be generated to produce sleep. Why isn't the "amount" of inhibition limited to that which would hold the impulse in check, without cutting down the activities of consciousness? But biological reactions are not always measured by need, and inhibition may overshoot the mark. This is especially understandable in man: the soldier, for example, is acutely aware of the need to stand fast, and of what will happen to him if he doesn't. The generation of a surplus of inhibition under these circumstances is analogous to what firemen do in putting out a fire: they often use more water than they need to.

A telling and decisive incident, one that will dispel any doubt on the importance of conditioned inhibition as a cause of sleepiness, occurred in the case of a healthy soldier who was posted as a sentinel one night from 2 to 4 a.m. (He was not one of the 25 subjects of this study.) At 4 o'clock the corporal of the guard failed to show up with the expected relief. Thinking the corporal would arrive at any moment, the sentinel walked his post patiently. Gradually, however, it dawned on him that the corporal must have fallen asleep, and he became more and more angry. He wanted to leave his post and denounce the corporal, but as a good soldier he successfully resisted this impulse. (The incident occurred in the United States, so there was no question of danger from the enemy.) Presently he realized he was getting tired and sleepy, and after a time it required the last ounce of self-discipline to urge his feet onward. There was a point in his tour where he had to climb twenty steps, and here his feet felt like lead. Finally, at 6 a.m., when he felt he couldn't possibly stay awake any longer, relief came at last. Now a remarkable thing happened: the fatigue and sleepiness, which a moment before had been overpowering, vanished in the wink of an eye, and the soldier left his post fresh and alert, as if nothing had happened.

The value of this incident lies in the fact that the fatigue and sleepiness vanished so suddenly on the arrival of relief. Having walked post from 2 to 4 a.m., the man was forced by circumstance to walk an extra two hours till 6. Could his fatigue and sleepiness have been due to the additional muscular effort put forth at a time when he was already tired? Not at all, for the ill effects of muscular overexertion don't clear up so suddenly without a substantial period of rest. They were due, rather, to conditioned inhibition (for he was suppressing the impulse to desert his post). They vanished in a trice the moment suppression became unnecessary (for, having been relieved, he could now come and go without

let). The dramatic recovery on the sudden lifting of the need for suppression can have only one meaning, and it endows this incident with the precision and decisiveness of a laboratory experiment.

*Differentiation*

Pavlov studied the ability to differentiate stimuli. He found, for example, that if a musical tone of 800 double vibrations is always followed by meat, the animal will salivate in response to neighboring tones as well. If then one gives meat only after the tone of 800 d. v. and never after the others, it is possible, within limits, to restrict the reflex response to the 800. To show how minutely some animals can differentiate tones, it may be noted that some dogs in Pavlov's laboratory could differentiate the tones of 800 and 812 d. v. Pavlov proved that differentiation is based on inhibition; in other words, the neighboring tones continue to evoke excitation in salivary centers, as they did earlier in the experiment prior to the achievement of differentiation, but they now evoke also an inhibitory process which holds the excitations in check. Moreover some animals respond to these inhibitory stimuli (the unreinforced neighboring tones) by falling asleep.

Again the experimental findings apply also to man. The notorious sleepiness of automobile drivers is not due to the monotony of the task, but to the continual differentiation of positive and negative stimuli which driving entails, with the consequent accumulation of inhibition.[5] The sentinel, especially when near the enemy, illustrates this well. Hearing a faint rustling noise out front, the sentinel is alerted and tensed for action; a moment later he may realize the noise was only the wind in the trees. His tension relaxes—excitations that were about to materialize in overt action are inhibited. Let this happen a dozen times in the night and the cumulative effect of many small "amounts" of inhibition may overwhelm him with sleepiness.

From the foregoing it is evident that the sleepiness of the sentinel cannot be dismissed as merely the effect of boredom. It rather is the effect of cumulative inhibition —both the inhibition that comes into play when he suppresses the wish to leave his post (conditioned inhibition), and that which is evoked when he differentiates positive and negative stimuli.

Whether or not a given "amount" of inhibition will produce sleepiness depends on how much inhibition there was already present to start with—another way of saying that a sentinel who has rested well before going on duty will be the better able to tolerate the inhibition that his task will evoke in him. It depends too on his constitutional susceptibility to inhibition. Pavlov found that animals vary in this respect, some falling asleep in a given set of circumstances while others don't. Using the term "inhibitability" (in analogy with excitability), one may say that the basis of narcolepsy lies in excessive inhibitability of the cerebral cortex.

In the foregoing analysis of the factors which promote somnolence in the sentinel, no mention has been made of motivation. The analysis applies to soldiers of good motivation, men who want to do their duty and want to stay awake. The problem is much more complex when poor motivation is added to the picture, as in the case of psychopaths who don't care whether they fall asleep, and who may even wish to do so in order thereby to give free rein to their insubordination and hostility.

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# Twelve-year Resume In A Clinic for Epilepsy

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An intensive study upon epilepsy was commenced in 1935* the results of which were incorporated in a report published by Northwestern University Medical School in 1936.[1] The stimulus afforded by this study led to the establishment of a clinic for epilepsy in the Montgomery Ward Clinics of Northwestern University Medical School in 1935. Since that time and until January 1948, we have treated 543 patients suffering from various convulsive disorders. Of these, in 311 we have had the opportunity of observing the patients for a sufficiently long time to make them useful for an analysis of the result of treatment. In addition certain interest is attached to 17 other patients who died and 23 who were transferred to institutions. (Table 1.)

TABLE 1

| | |
|---|---|
| Former patients treated sufficiently long | 223 |
| Current patients treated sufficiently long | 88 |
| Former patients with insufficient length of treatment | 57 |
| Recent patients with insufficient length of treatment | 34 |
| Institutionalized | 23 |
| Died | 17 |
| Total up to January 1, 1948 | 543 |

The evaluation of the efficacy of an anticonvulsant drug depends upon criteria concerning which there is no uniformity of opinion. Until such uniformity of opinion is evolved it is impossible to compare various clinical results with any degree of accuracy or certainty. While some observers hold that when a drug diminishes the number or severity of attacks it is efficacious, we feel that the only criterion of efficacy is the prolonged cessation of all attacks (a long remission) or the complete absence of attacks up to the time of a report (a terminal remission).

Many difficulties present themselves in the evaluation of the efficacy of the treatment of epilepsy in an outpatient department of a medical school. A number of patients not finding immediate relief change loyalty from one clinic to another; others are neglectful; some because of change in economic status consult private physicians; still others having their attacks stopped, continue medication without reporting to the clinic. At any rate a large number of patients who may have presented themselves to our clinic some years ago have discontinued their visits.

We are forced, therefore, to confine our study to patients currently attending the clinic and to the records of those who have discontinued their visits and whose records before their defection may be followed over a significantly long period of time.

It has been the custom in the clinic for convulsive disorders, to evaluate the patient's history with respect to previous therapy if any. We have never taken the patient's word as to his being a patient recalcitrant to treatment. If the patient was not in a remission at the time seen, he was usually treated by sodium bromide. The dose given was sufficient to give a blood bromide level of approximately 200 mg. per cent. If a remission did not occur on this therapy after an adequate trial, usually a few months, he would then be tried on phenobarbital in doses from 2 to 6 grains per day. If, after a suitable period, a remission did not occur, a combination of sodium bromide and phenobarbital was given. The majority of patients would usually have a remission on such a regime. If this did not occur the patient was placed upon dilantin, alone or in combination with one of the aforementioned drugs. or on mesantoin, or on newer drugs being tried in the clinic. To classify a patient as recalcitrant requires many months to years of careful supervision. It has been our experience that patients so classified as recalcitrant after a long period of therapy will do very little better with one than with other therapies. Our observations on recalcitrant patients treated with dilantin and mesantoin have been published previously in reports of smaller groups of patients.[2,3]

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*Effect of Therapy on Remissions*

In 118 or 83.02 per cent of 142 cases treated only by sodium bromide, all attacks of any form were stopped for periods varying from six months to two years and longer preceding their last report to the Clinic. These are classified as terminal remissions; some patients having no recurrence for 10 years or more.

In 20 other patients, attacks were stopped for periods varying from three months to one year, returning for one or another reason, then again to disappear, but for a time less than six months prior to their last report. These are classified as long remissions interspersed with exacerbations and were brought about in 14.08 per cent of the 142 cases. In 4 of the 142 cases or 2.9 per cent no long remissions were brought about.

Of 91 patients treated only by phenobarbital, in 73 or 80 per cent terminal remissions were brought about. In the remaining 18, long remissions and exacerbations resulted.

Of 25 cases treated by both phenobarbital and bromides no terminal remission was seen, and in 12 or 48 per cent long remissions with exacerbation occurred.

Of 5 patients treated alone by dilantin, in none was a terminal remission brought about, but in all, long remissions then exacerbations occurred.

Of 12 patients treated by dilantin and phenobarbital, in 4 or 33 per cent, terminal remissions were brought about, and in 8 or 66 per cent, long remissions and exacerbations occurred.

Of 17 treated by a combination of dilantin, bromides, and phenobarbital, no terminal remissions were brought about, and long remissions and exacerbations occurred in 6 or 32 per cent.

Of 19 treated by mesantoin and phenobarbital, terminal remissions were brought about in 7 or 36 per cent, and long remissions and exacerbations occurred in 12 or 63 per cent.

In resumé, in 191 patients treated by sodium bromide and/or phenobarbital, a terminal remission of all spells resulted. This is 73 per cent of 258 patients treated with these drugs alone or 61 per cent of the grand total of 311 patients treated with all drugs. When the remissions by other drugs are added, the terminal remission increases from 191 or 61 per cent to 202 or 68 per cent of the grand total. Again the addition of all drugs raised the remission and exacerbation rate *per se* very little from 50 patients or 16 per cent to 72 or 23 per cent of the grand total.

It should not be construed that dilantin or mesantoin are not used in more patients than shown. As mentioned previously, it was our custom to treat new patients with sodium bromide, then phenobarbital alone or in combination with sodium bromide. If this method was unsuccessful, the newer drugs were used in attempt to get a remission of all spells. Thus, in this group 258 patients were treated with sodium bromide alone or phenobarbital alone or both in combination; and 53 patients were treated with dilantin, mesantoin, each alone or in combination with phenobarbital, and sodium bromide. The results of therapy in larger groups of patients treated with dilantin alone and mesantoin has been previously published.[2,3] Furthermore, in a more recent survey of the results of therapy, an analysis was made of 115 patients currently treated up to January, 1950. In this group only 14 patients were treated with sodium bromide, and in 11 or 80 per cent a terminal remission occurred from 4 months up to 10 years. (In this older report 82 per cent of 142 patients had a remission.) In 48 patients treated with phenobarbital, there were 43 or 90 per cent with a remission of all spells from 4 months to 15 years. (In this older group 80 per cent of 91 patients had a remission.) In 53 patients treated with newer drugs such as dilantin, mesantoin, thiantoin, phenurone, tridione, and paradione, most of them in combination with phenobarbital or sodium bromide, there were 24 or 45 per cent having a remission of from 4 months to 5 years compared to the older group of 53 patients with 11 or 20 per cent having a remission.

It is interesting, however, in the totals of older and current groups that the total remission rate is the same, or 68 per cent. The newer drugs have then increased only slightly the total remission rate, but have allowed a greater selection of some less sed-

ative drugs. It would seem also that a remission can be obtained by any of the anticonvulsant drugs if adequately used in about 60 per cent of the general run of patients suffering from epilepsy.

*Analysis by Type of Seizure*

In 136 patients only major or grand mal seizures occurred. In these a terminal remission occurred in 104 or 76 per cent, and in 27 additional patients remissions and exacerbations occurred, or a total of 131 or 97 per cent had terminal remissions plus remissions and exacerbations.

In 91 patients major and minor seizures occurred, and in 51 or 56 per cent a remission was obtained. In 81 or 89 per cent, terminal remissions and exacerbations occurred.

In 81 patients major and/or minor and/or equivalent seizures occurred and in 45 or 55 per cent a terminal remission occurred. In 63 or 77 per cent remissions plus remission and exacerbations were obtained.

In only 3 patients did we have minor seizures alone, and in 2 or 66 per cent did we get a terminal remission.

Thus, major seizures alone offer the best prognosis giving a remission rate of 76 per cent in contrast to 55 per cent when the patient has minor or equivalent seizures in addition.

*Type of Convulsive Disorder and Remission Rate*

Patients with idiopathic or symptomatic (organic) epilepsy responded equally well to treatment, 65 and 70 per cent respectively. However, in those patients classified as focal epilepsy by virtue of a focal aura or convulsion starting from a focus, the remission rate was less (55 per cent). This may well be due to the focal group's having a high incidence of equivalent seizures which respond less well to therapy.

*Analysis of Organic or Symptomatic Epileptic Patients*

The cause of the organic epilepsy in 82 patients was post infectious in 27, chronic alcoholism in 3, cerebral trauma in 19, cerebrovascular apoplexy in 13, birth trauma in 8, brain tumor in 8, central nervous system syphilis in 5, and toxic state in 2.

It was noted that recalcitrant patients as far as therapy was found in only two out of 24 cases of post infectious states, in 3 of 19 cases of brain injury, in two of 13 cases following cerebral apoplexy and 2 out of 5 patients with central nervous system syphilis. Syphilis and brain trauma showed the highest percentage of recalcitrant patients.

*Analysis of Institutional Patients*

The total number of patients institutionalized was 23.

Only 8, or approximately one-third, were so-called idiopathic, and 6 were suffering from organic brain diseases exclusive of mental deficiency. Likewise, only 7 or 31 per cent had only major attacks.

Of the 23 patients, 5 had to be sent to an epileptic colony because of their more important intellectual defect. Fourteen were committed to a mental institution because of their dementia mainly or their mental disorder due to or associated with their epilepsy. In 6 of these patients the mental condition may have been a definite part of the epilepsy *per se* such as post epileptic psychoses, behavior disorders (possible psychomotor attacks).

A remission of all spells occurred in 4 of the 23 patients treated, but their mental or behavior disorders still necessitated incarceration.

*Deceased Patients*

There were 17 known dead. In 7, the cause was due to or associated with the epilepsy, i.e., in 3, brain tumor, 2 committed suicide, 1 died in status epilepticus, and in one a toxic hepatitis developed presumably due to drug therapy. Actually only one of the patients died as a result of epilepsy (status).

Twelve out of the 17 patients died under 50 years of age, which is rather young considering life expectancy.

Only two patients of this group had a remission of spells before death.

*Summary and Conclusion*

1. It is difficult to evaluate the efficacy of a drug unless it produces prolonged cessation of attacks.
2. In an analysis of 311 patients a remission of 6 months up to 10 years was pro-

duced in 202 or 68 per cent by the use of anticonvulsant drugs.

3. The older drugs such as sodium bromide alone or phenobarbital alone or in combination with sodium bromide produced a remission in 61 per cent of the whole group. The addition of dilantin and mesantoin each alone or in combination with phenobarbital and sodium bromide increased the remission rate to about 68 per cent. In a recent analysis of 115 patients treated up to January 1, 1950, in which fewer patients were treated with drugs such as dilantin, mesantoin, thiantoin, phenurone, paradione, triodine, the total remission rate is still 68 per cent.
4. The remission of all spells was highest, 76 per cent, when only major seizures were present.
5. The presence of minor or equivalent seizures decreased the remission rate from 76 to 55 and 56 per cent.
6. The remission rate was very similar in idiopathic and symptomatic epilepsy. The presence of focal attacks decreased the remission rate.
7. Remissions appeared to be more difficult to attain in central nervous system syphilis compared to other etiologic factors.
8. The deceased patients died at an earlier age than expected, but only one of the 17 died as a result of epilepsy.
9. Of the whole group, 23 patients were institutionalized because of mental deficiency, epilepsy, or organic brain disease.

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# Neurological Clinical Pathological Conference

of the

Cincinnati General Hospital

CHARLES D. ARING, M.D., *Editor*

Case No. 261154

## *Presentation*

A six-year-old colored girl was well until March 18, 1950 when she became ill with fever, anorexia, and a papular eruption in the scalp. She had had none of the childhood diseases other than measles at the age of 2 years. On the day following the onset of the present illness the eruption appeared on her face and trunk. Although she felt fairly well, her fever continued. By March 20 the papules had become encrusted and during the evening of that day she developed bitemporal headache which persisted through the night and she slept only intermittently. By 5:30 A.M. of March 21 the headache had become severe and she had a generalized convulsion, following which she remained unconscious and was cyanotic.

When first examined at the hospital at 7:00 A.M., March 21 she was having continuous generalized convulsions and was cyanotic. Her body frequently assumed opisthotonic position associated with rapid, noisy, labored breathing. Her temperature was 106.4 degrees F. and her pulse 160 per minute. There were numerous encrusted cutaneous papules distributed over the head, trunk and extremities. There was moderate cervical lymphadenopathy. Her pupils were dilated and did not react to light. The optic discs and tympanic membranes were injected. Many rales were heard throughout both lungs. There was no nuchal rigidity, and Kernig's and Brudzinski's signs were not present. The plantar reflexes were extensor bilaterally.

Initial laboratory studies showed normal urine, a hemoglobin of 16.5 grams per cent, 6.2 million red cells per cmm., 30,400 white cells per cmm. in the peripheral blood with a differential white count of 90 per cent polymorphonuclear leukocytes, 8 per cent lymphocytes and 2 per cent eosinophiles. The cerebrospinal fluid had an initial pressure of 440 mm. of water; 12 cc. of cloudy fluid were removed and the final pressure was 270 mm. of water. The fluid contained 860 white blood cells per cmm. of which 32 per cent were lymphocytes and 68 per cent polymorphonuclear leukocytes. There were 175 mg. per cent of protein, 122 mg. per cent of sugar and 708 mg. per cent of chlorides in the cerebrospinal fluid. No organisms were present on Gram stain of the sediment.

The convulsions could be controlled only imperfectly with paraldehyde and barbiturates and clonic

movements persisted despite large doses of these drugs. Therapy consisted of general supportive measures, intravenous fluids, sulfadiazine, penicillin and streptomycin. Although the temperature fell to the 100 to 101 degrees F. range on March 22, the patient never regained consciousness. Her heart rate continued at 120 to 140 per minute. She passed several dark liquid stools and vomited coffee grounds emesis on the 22nd. During the evening of March 22 abdominal distention developed and the clonic movements stopped. Her respirations which had been quite rapid throughout became shallow and she expired at 7:55 A.M. on March 23.

### *Differential Diagnosis*

*Dr. Clifford McIntyre:* This particular case is most interesting as it includes a pathological process in the skin as well as in the central nervous system, the disease of the skin being reflected in changes in neural tissue. The bridge between neurology and dermatology is being traversed more frequently as we become more curious about the nature of the simultaneous or sequential disorder of skin and nervous system.

This child fell ill of a skin disease which is delineated as a papular eruption of the head region, spreading to the neck and trunk. It probably was apparent to the physician who first observed the child that she was suffering from one of the exanthemata. The head is a characteristic place for many of these diseases to begin; variola, varicella, and rubeola may begin on the face and spread to the trunk and extremities. The severity of the symptoms would certainly suggest variola for the not unusual course of this disease in children is that of a fulminating infection with many constitutional symptoms. The biphasic feature of this disease, namely the subsidence of symptoms and exacerbation after a day of rest, is present in this case. Chicken pox is accompanied by constitutional disturbances such as fever, headache, and anorexia. Were rubeola to be considered, one would have to presuppose that Koplik spots and oral signs had been overlooked and that the rash was atypical, being papular rather than macular or maculopapular.

Other papular rashes should be reviewed, namely pyoderma including impetigo and streptoderma, syphilis, eczema vaccinum, generalized vaccinia, and there are other more obscure causes of this type of eruption. Impetigo is found in the head region, but would be unlikely to spread in the fashion described. Pyoderma and vaccinia are spread by the child scratching various areas of the body. One could postulate that the infective agent of pyoderma, either streptococcus or staphlococcus, had been carried by the veins of the nasal region to the cavernous sinus and thence to the meninges. Such an infection would most likely be modified by antibiotic therapy and could make itself known upon examination of the stained cerebrospinal fluid. There was no external ophthalmophegia, choked disc, or other sign of cavernous sinus infection and thrombosis to support this suspicion. Vaccinia itself is a mild disease caused by vaccination or contact with an open vaccinated area on another individual. Such might happen to a child playing with other children recently vaccinated. The severe neurological sequellae of vaccinia are due to an acute disseminated encephalomyelitis which attacks white matter somewhat more than gray matter, and shows a specificity for neural tissue surrounding blood vessels. The process is a demyelinizing one. These cases occur anywhere from 1:20,000 to 1:3,000 vaccinations.

Eczema vaccinum is a disease which occurs in children liable to atopic eruptions and allergic disturbances, who are vaccinated and then develop a macular or even papular rash as an allergic response to the vaccinia which has been introduced. No severe neurological sequellae have been reported in this disease to my knowledge. The spread of a papular syphilid is seldom as rapid as that seen in this child. It would be unlikely to see a concomitant severe luetic meningitis and severe skin involvement, relying upon that old dermatological maxim that these do not occur in the same individual. This rule has its exceptions; however, the exception does not validate the diagnosis of syphilis in this case. The Wassermann, which we may presume to have been negative, would have indicated a luetic process in the cerebrospinal fluid and the response to penicillin should have been obvious.

Scarlatina usually produces a macular rash, occasionally a pin point papular rash, but it exfoliates rather than becoming en-

crusted. Weil's disease may be accompanied by a papular rash and, although this entity is endemic in the Cincinnati area, its development is usually marked by the appearance of jaundice and yellow cerebrospinal fluid. Sporotrichosis caused by sporotrichum schenki, a fungus, also produces a papular eruption, but a mycotic infestation of the nervous system would have been apparent upon microscopic inspection of the cerebrospinal fluid.

There is one neurotropic virus which produces a skin eruption which should be seriously considered. Herpes zoster appears upon the head when it strikes the Gasserian ganglion. In rare cases, it becomes generalized and attacks the entire chain of posterior root and sensory ganglia. It may begin as a papule, never reach the state of a vesicle, but become encrusted as did the rash in this child. When a cerebrospinal fluid examination is done in herpes zoster, it often reveals pleocytosis, for the inflammation of the sensory ganglia spills over into fluid. At times this pleocytosis may assume the proportion of a severe meningitis caused by the virus of herpes zoster. Encephalitis due to herpes zoster has also been reported.

The neurological signs and symptoms which developed in this child are definitive and point to the portions of the central nervous system involved. There were generalized convulsions, opisthotonos, dilated fixed pupils, but no meningeal signs. These would indicate that the cerebral cortex and the midbrain area were mainly involved. The opisthotonos and pupillary findings indicate that midbrain structures have been released from higher control and that a modified decerebrate rigidity has occurred. This may have resulted either from direct inflammation of the midbrain or pressure upon it, the increased intracranial pressure damaging the cerebral peduncles.

In spite of the pleocytosis, this is not the picture of a meningitis, but rather that of encephalitis, cellular elements of inflammation from the brain substance spilling into the cerebrospinal fluid via the Virchow-Robin spaces which are the vascular entrances and exits to and from the neural tissue. The cerebrospinal fluid also indicates brain swelling or inflammation of the leptomeninges and I am inclined to favor the former as the cause of the increased pressure and protein content. The hyperglycorrhachia of 122 mg. per cent would militate against a pyogenic infection such as meningococcus meningitis, which also produces a rash, perhaps pustular, but characteristically petechial. The normal chloride content and fulminating course of the disease cause me to dismiss tuberculous meningitis.

A rash may be found in either encephalitis type A or B, but is not characteristic. The diagnosis of encephalitis type A in this decade is rarely made and is recently a more protracted disease than the one described. A type B encephalitis would also be unlikely in this area at this time unless the patient had rather intimate contact with infected laboratory animals. Rickettsial infections such as spotted fever or rickettsial pox which are tick-borne would be most unusual in the month of March; neither would the neurological sequellae be of this character.

A neurotropic virus is implicated as the cause of the child's illness. This virus may be the same virus which has been dermatropic, or it may bear a relationship to the virus producing the pustule which might be termed commensal. The outbreak of the dermatrophic infection may have produced the neurotropic virus efflorescence. It has been argued long that varicella and herpes zoster are either mutants of the same virus or separate viruses. They bear some relationship to each other because of epidemiological evidence, inconclusive as it is, that the one disease may precede the other in two individuals who have had contact with each other. Dermatologists find that when the two diseases occur side by side, varicella in a child, and herpes zoster in an oldster, they are indistinguishable by inspection. This child suffered from an encephalitis which might have been caused by the interrelated varicella-herpes zoster complex.

This particular complication of varicella is rare but not unknown. Neurological symptoms tend to develop on the fifth day of the rash. There is little uniformity of the clinical types of the disease, for encephalitis,

myelitic, ataxic, and meningitic forms have been reported. Basal ganglia disturbances, choreic movements, dysphagia, and mental disorder also have been reported. From the neuropathological standpoint, the disease has been explored in few cases, but according to one investigator, it differs from the other acute disseminated encephalomyelidites in that there is no perivascular demyelinization, presumably due to the fact that the varicella virus has no selective action upon myelin sheaths. There does, however, appear to be neuronolysis and perivascular inflammation occurs both in gray and white matter. The involvement may be of the cerebellum as well as of cerebrum and basilar structures. All in all, few cases have been studied satisfactorily.

*Dr. Paul Wozencraft:* This patient came to autopsy six hours post mortem. The brain weighed 1380 grams (the average normal brain weight for a child of this age is 1240 grams) and was edematous and showed petechial hemorrhages. While there were visceral changes of interest, they are not specific. There was necrosis of the splenic follicles of severity comparable to that of patients dying of diphtheria. A moderately severe generalized necrosis of the liver was present. There was also a severe interstitial pneumonitis with areas of bronchopneumonia.

*Dr. Albert B. Sabin:* This patient became ill on March 18, 1950 with an eruption which left little doubt in the minds of the physicians who saw her that it was characteristic of chicken pox. On March 20 the skin lesions had already crusted and next day she developed severe headache and towards the end of the day, convulsions. The cerebrospinal fluid exhibited a predominantly mononuclear leucocytic pleocytosis. The patient died early in the morning on March 23, just five days after appearance of the rash.

We were presented here with an opportunity to obtain additional information on the effect of varicella virus on the nervous system, particularly in relation to the question of its identity with the virus of herpes zoster. Neither the varicella virus nor the herpes zoster virus have as yet been transmitted to experimental animals and we know very little about their basic properties. I will not review all the observations which have led some to suspect that varicella and herpes zoster may perhaps be manifestations of a single virus.[1] However, since such a possibility deserves serious consideration and because the manifestations of herpes zoster suggest particular involvement of certain neurones, the question naturally arises whether in a patient who died very early after onset of varicella, one might find any evidence of specific neuronal damage directly attributable to varicella virus. I was acutely interested in finding out whether it would be possible by an examination of the sensory ganglia—the Gasserian, cervical, thoracic and lumbar—to find changes in the neurones which might suggest that they were invaded by the varicella virus. If this property could be demonstrated for varicella virus, it would not be unreasonable to suppose that the virus could remain dormant in the sensory ganglia, until some incident, capable of upsetting this host-virus equilibrium, started the virus moving centripetally and centrifugally along insulated neural pathways to produce the clinical manifestations which constitute the syndrome of herpes zoster.

It is common knowledge that acidophilic, intranuclear inclusions have been seen in the epithelial cells in the skin lesions of varicella and herpes zoster. Although very few necropsies on patients dying of varicella have been reported without involvement of the nervous system, there are at least two reports on fatal cases in infants, in which widespread visceral necrosis was associated with the same type of acidophilic intranuclear inclusions.[2,3] Since the present patient died so soon after onset, it was of particular interest to determine whether or not similar intranuclear inclusions might be found in the neurones. Furthermore, since newborn mice had not to my knowledge been tested for susceptibility to the varicella virus, I thought it desirable to carry out such tests with the cerebrospinal fluid (obtained one day before death) and the brain tissue of this patient. These tests in mice were also desirable to determine whether or not the virus of *herpes simplex* was present in the nervous system of this patient.

Now, let me summarize what was found.

To begin with the tests in mice (which included several one to two-day old litters as well as older animals) yielded no virus. The autopsy was done six hours after death and the viscera and nervous system were well preserved. The tissues, in thin slices, were fixed in Zenker's fluid containing 5 per cent acetic acid, to permit optimum demonstration of the acidophilic intranuclear inclusions. The sections were stained with eosin-methylene blue. However, despite a careful search, intranuclear inclusions were found neither in the skin lesions, viscera nor anywhere in the nervous system. Serial sections through a crusted vesicle in the skin showed the focal necrosis of epithelial cells without suppuration or inflammation in the subepithelial structures, which is compatible with the changes one might expect in varicella. The absence of intranuclear inclusions in the surrounding epithelial cells could either be due to the fact that five days had elapsed since the first appearance of the lesion or that these inclusions are less common in older children than in infants. Although the lungs were the seat of an interstitial pneumonitis, with focal subpleural hemorrhages and intra-alveolar edema, there were no specific lesions or inflammatory changes in the trachea or bronchi. The portions of liver which I had examined revealed no foci of necrosis. Cellular infiltration was also absent except in an occasional portal space which is of doubtful significance. The presence of large numbers of mitotic figures in the nuclei of the liver cells may perhaps be the result of the action of varicella virus on them. Since the portion of the liver examined by Dr. Wozencraft exhibited foci of necrosis, it would appear that the liver was affected although in a spotty fashion. Although the spleen showed considerable distortion of its normal architecture, it did not exhibit the extensive necrotic lesions on finds in generalized vaccinia or variola. No areas of necrosis were found in the kidneys, adrenals, pancreas or thymus. One of the ovaries, however, exhibited an area of acute acidophilic necrosis, affecting the wall of one of the multiple cysts which were seen grossly.

The reason one is so interested in the visceral involvement by the virus of varicella is that experimental studies on other viruses which affect both the viscera and the nervous system, e.g., herpes simplex, pseudorabies and B virus, have revealed that the viscera can be the site from which the virus then invades the sensory ganglia via neural pathways.

In the nervous system, the meninges showed very sparse focal infiltration with mononuclear cells. Many of the sections revealed no meningeal infiltration at all. One certainly cannot speak here of a meningitis. The sparse infiltration is most likely the result of an overflow of cells from the Virchow-Robin spaces of some of the blood vessels. Throughout the cerebral cortex, medulla and spinal cord, it was possible to find blood vessels, which exhibited this perivascular infiltration in the Virchow-Robin spaces, predominantly in the gray matter or at the junction of the gray and white matter. The cellular infiltration extended into the surrounding parachymal tissue only occasionally, and the eosin-methylene blue stained sections revealed no lesions which could be interpreted as perivascular demyelinization, although an occasional focus with general loss of ground substance was seen especially at the junction of the gray and white matter in the spinal cord. The neurones in the spinal cord, medulla and cerebellum (including the Purkinje's cells) were remarkably well preserved. However, the most striking pathological change in the nervous system of this patient was found in almost all the neurones of the cerebral cortex—frontal, parietal, temporal and occipital. The change can best be described as an acute necrosis in which the cytoplasm loses the Nissl substance and becomes homogeneously "smooth" and acidophilic, taking the red eosin stain, and the nucleus loses its normal architecture exhibiting extreme pyknosis and karyorrhexis. It is noteworthy that the surrounding glia, blood vessels and other structures stain normally indicating that the fixation and staining were good, and that the neuronal damage was somehow selective. This type of cortical neuronal damage was first described in varicella encephalitis by Zimmerman and Yannet[4] in 1931 and in pertussis encephalopathy by Hussler and Spatz[5] in 1924. This type of

neuronal damage, sometimes also called ischemic necrosis, has been encountered in certain cases of toxic encephalopathy.

Since I needed some personal convincing that this widespread neuronal damage was neither an artifact resulting from poor fixation and staining nor so nonspecific that it would be encountered in any patient with encephalitis, I examined similar sections of the nervous system, fixed and stained in an identical manner, from several patients who died of: a) Japanese B encephalitis, b) encephalomyelitis due to B virus of monkey origin, and c) postvaccinial encephalomyelitis. In none of these did I find this type of cortical neuronal damage. In Japanese B encephalitis, as in other specific viral encephalitic lesions, there is focal destruction of neurones with neuronophagia and inflammatory reaction, but the surrounding neurones stain in normal fashion. I cannot escape the conclusion that the widespread cortical neuronal damage in this patient is the most important lesion, although the mechanism of its production is a mystery. Furthermore, for the reasons mentioned above, I cannot regard it as being due to a direct attack of the varicella virus on the cortical neurones. In no instance was anything found even remotely suggesting an acidophilic or other type of intranuclear inclusion.

An examination of almost all the cervical, thoracic and lumbar spinal ganglia revealed neither neuronal damage nor any inflammatory reaction. However, both Gasserian ganglia showed extensive interstitial, focal lymphocytic infiltration. The important question, however, was whether any evidence could be found that the neurones were specifically attacked or destroyed with consequent neuronophagia and interstitial cellular infiltration. The neurones, however, appeared intact with no intranuclear inclusions, although by trying very hard I might have convinced myself that occasional aggregates of mononuclear cells and lymphocytes might represent foci of neuronophagia. Now it is quite possible that at one time I might have accepted these lesions as indicative of invasion of the Gasserian ganglia by the varicella virus. I might also have imagined that it was pure chance that in this patient only the Gasserian ganglia were involved and that in other patients, certain of the spinal ganglia could be similarly involved. Thus, one could have histological evidence that the varicella virus can invade the sensory ganglia, and under certain conditions give rise to the syndrome of herpes zoster.

However, I must now proceed to tell you why I cannot accept these lesions in the Gasserian ganglia as indicative of invasion by the varicella virus. In a study of the significance of certain cellular infiltrative changes in sensory and sympathetic ganglia, Bodian and Howe[6] recently found that a certain number of uninoculated chimpanzees exhibited rather severe mononuclear infiltrative lesions in the trigeminal and sympathetic ganglia. In one chimpanzee, the stellate and superior cervical sympathetic ganglia exhibited germinal centers characteristic of lymphatic nodules in the center of large mononuclear infiltrative lesions. I did not know that lymphoid tissue may be intimately associated with the Gasserian ganglia, but the section through one of the Gasserian ganglia of the present patient showed a small, distinct lymph node in the connective tissue surrounding the whole complex. It is not improbable, therefore, that the mononuclear infiltrates in the Gasserian ganglia may represent extensions of lymphoid channels rather than neuronophagia or inflammatory reaction to damaged neurons. At any rate, I must conclude, that no historical evidence was obtained of invasion of the Gasserian or other sensory ganglia by varicella virus. That is not to say that such invasion may not occur without demonstrable histologic change, but the concept of its occurrence must remain an unproved assumption.

In conclusion, then, we must say that no evidence was found of invasion of the nervous system by the varicella virus. The scattered perivascular infiltrative changes in the nervous system of this patient are of the type seen in the postinfectious type of encephalitis with or without demyelinization. However, the most striking pathologic change is the ischemic type of necrosis which affected almost all the cortical neurones.

*Clinical Diagnosis*

Varicella encephalitis.

*Dr. McIntyre's Diagnosis*

Varicella encephalitis.

*Anatomical Diagnosis*

Varicella encephalitis.
Interstitial pneumonitis.
Spotty focal necrosis of the liver.

*Dr. J. Park Biehl:* Dr. Savin, would you conclude that these changes in the central nervous system are on an anoxic basis?

*Dr. Sabin:* I don't see how we can tell. All I would say is that by some unknown mechanism, the cortical neurones were selectively damaged to produce a picture described by some as "ischemic necrosis." Is there anyone here who can tell us just what causes ischemic necrosis? Although the term itself suggests lack of blood supply, I personally do not know. There are many things that might cause acute death of nerve cells. I think the interpretation that these are dead cortical neurones is justified. One may speculate, for example, whether it is possible that part of this process involves a diminution in the blood supply of certain areas of the cortex. It might be interesting to test the effect of antihistaminic compounds next time one gets a patient with varicella encephalitis. I understand that antihistaminics have been tried with some success in patients presenting certain types of convulsions.

*Dr. Biehl:* Would the perivascular cuffing be secondary?

*Dr. Sabin:* The cause of perivascular cuffing in postinfectious encephalitis is unknown. Whether it is due to a reaction across that particular blood vessel between antibrain antibody and antigen in the brain tissue, can only be hypothesized. However, I doubt that it is a reaction to the extensive neuronal damage in this case. First, because many areas which exhibited the neuronal necrosis showed no perivascular infiltration, and, secondly, because in other types of "toxic" encephalopathy or in pertussis, similar neuronal necrosis is not accompanied by perivascular infiltration.

*Dr. Biehl:* Would you separate the two?

*Dr. Sabin:* It is possible that two distinct mechanisms may be operating here, side by side. A little earlier, I mentioned a patient with postvaccinial encephalomyelitis who died within 24 hours after the first convulsion. Dr. Aring and I studied this case in 1942, and I remember that despite an extensive search in many myelin-stained sections, no areas of demyelinization were found. All that was noted were some areas of perivascular infiltration. I think that Ferraro has reported certain cases of postvaccinial and postinfectious encephalomyelitis exhibiting only perivascular infiltration with demyelinization. The patient with postvaccinial encephalomyelitis whose sections I restudied did not exhibit these acute neuronal changes. So you can have perivascular infiltration without the neuronal necrosis. In this patient with postvaricella encephalitis we apparently had both.

*Dr. Daniel Osher:* Dr. Sabin, aside from these cortical cell changes, there were widespread acute perivascular changes?

*Dr. Sabin:* Yes, widespread but not abundant.

*Dr. Osher:* Isn't it rather early to expect much in the way of demyelinization if this is an allergic response?

*Dr. Sabin:* Zimmerman and Yannet[4] reported perivascular demyelinization in a case of varicella encephalitis in which death occurred within 24 hours after onset of convulsions.

*Student:* Do you ever see such changes with hyperpyria or associated with any other state?

*Dr. Sabin:* This extensive neuronal damage has been seen in instances described as "toxic encephalopathy" and in pertussis encephalopathy. I was unable to find similar changes in patients dying with virus encephalomyelitis. Therefore, it isn't necessarily associated with high fever and convulsions.

*Student:* What about the neurones in patients who die in status epilepticus? Do they ever show any changes like this?

*Dr. Chas. D. Aring:* No.. Patients dying after a series of convulsions typically have changes in the area of the Ammon's horn and not generalized throughout the cortical layers.

*Dr. Sabin:* I might say that in this patient the neurones in Ammon's horn, particularly the smaller nerve cells, were well preserved.

*Dr. McIntyre:* Some of the few cases which have been described neuropathologically have lived fifteen days after the onset of varicella and one would expect that demyelinization would have appeared by that time. In no case has there been described perivascular infiltrations.

*Dr. Sabin:* I would want to check on that. Have you checked up on the individual autopsies?

*Dr. McIntyre:* No.

*Student:* Dr. Sabin, do you think that in chicken pox and other types of virus diseases the virus is present in the nervous system itself and can produce encephalomyelitis, rather than that all of these changes represent some post-infectious change?

*Dr. Sabin:* In recent years it has been found that in mumps infection it is possible to get two types of involvement of the nervous system. One is actually caused by the virus itself, which generally manifests itself as a true mumps meningitis, with the virus in the cerebrospinal fluid, but thus far there are no fatalities on record from this type of case. The other type of central nervous system involvement following mumps infection is more severe and at autopsy the post-infectious demyelinating type of pathologic change has been found. The possibility occurred, therefore, that perhaps also in varicella, there may be two types of central nervous system involvement, one due to the virus itself and the other as a "post-infectious" manifestation. I would say that at the present time

we have no evidence for direct involvement of the nervous system by varicella virus. It may be possible to investigate this in the future by a rather ingenious experimental procedure that has recently been introduced by Goodpasture and Anderson.[7] They showed that human skin can be grafted on the chorioallantoic membrane of a chick embryo, and that after inoculation with the vesical fluid from herpes zoster, typical changes, including acidophilic intranuclear inclusions, develop in the skin. That skin graft is a true graft because the chick erythrocytes invade the vascular channels in the human skin. Herpes simplex virus will grow directly on the chorioallantoic membrane, but the herpes zoster virus will grow only on the grafted human skin. This has not yet been done with varicella virus. As a matter of fact we have here a possibility of determining whether or not the varicella and zoster viruses are really closely related because it ought to be possible to carry out appropriate cross neutralization tests on grafted human skin. It might also be desirable to test cerebrospinal fluid and brain tissue from patients with varicella encephalitis on the graft skin. It is of interest that the first skin graft with which Goodpasture and Anderson got these beautiful results was derived from a 57-year-old lady who the year before had herpes zoster, indicating that the skin washed free of antibodies could still be infected with herpes zoster virus. The infectious zoster fluid which was used to perform this experiment came from a child who a year before had chicken pox.

*Dr. Biehl:* Will measles virus directly invade the nervous system?

*Dr. Sabin:* There is not enough evidence for such a conclusion. There was a suggestion in one instance that the brain tissue from a patient with measles encephalitis produced measles in a monkey.[8]

*Dr. Biehl:* Morphologically does the lesion appear to be due to virus?

*Dr. Sabin:* No, it is the post-infectious demyelinating type of change. In vaccinia also there is no evidence that post-vaccinial encephalomyelitis is caused by the virus itself. One cannot recover the virus from the nervous system. The lesions are of the same type as encountered in the encephalitis following measles and chicken pox, or after vaccination with killed rabies vaccine. The changes caused by the neuronotropic viruses themselves—and in that category are included herpes simplex, equine encephalitis, Japanese B encephalitis and others—are totally different from the manifestations that you see here. I would like while I'm talking about post-vaccinial encephalitis to ask Dr. McIntyre where he got his figures that it may occur once in every 20,000 vaccinations. I believe that there is something wrong there. The records of the army with regard to post-vaccinial encephalitis, indicate that among approximately 10,000,000 men, who were vaccinated against small pox during World War II, there were only four possible cases, including the one we studied here, of post-vaccinial encephalitis. There were reports years ago, particularly in Europe, of a higher incidence but for some reason in this country in recent years, it has been very low. I think that the same low incidence was encountered about two years ago when many of the population of New York City were suddenly vaccinated against small pox.

*Dr. McIntyre:* Those early reports come from Holland, which had a high incidence of the disease in the 1920's. It was remarked that the incidence in Great Britain and the United States was much lower than in Holland and Germany.

*Dr. Biehl:* Dr. Sabin, do you feel that in zoster the virus is in the ganglia and travels down the nerve and invades the skin?

*Dr. Sabin:* If I had to formulate a hypothesis of how zoster virus works, on the basis of clinical and pathological observations it would be something like this. The virus would enter the body either through the respiratory or alimentary tracts and localize at the portal of entry and in the viscera. From there it would spread by neural pathways to various sensory ganglia. From the sensory ganglia, it would spread centrifugally along the nerves of the corresponding dermatomes to the skin to produce the zoster lesion. In some or all instances it would spread centripetally to the central nervous system. Now, this image is based not only on the clinical and pathological picture of herpes zoster but also on the known behavior of certain other viruses which have been used in experimental animals. Thus, after intravenous injection of the viruses of pseudorabies, herpes simplex or B virus in rabbits, localization in the viscera occurs and from the viscera spreads to the corresponding sensory ganglia. With the B virus, I have been able recently to produce experimental zoster-like lesions in the rabbit. But since we cannot work experimentally with human herpes zoster, this remains a very hypothetical picture.

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